THE EFFECTS OF COMPRESSION AND THE ROLE OF THE HEMATOMA IN FRACTURE HEALING.

THE EFFECTS OF COMPRESSION AND THE ROLE OF THE HEMATOMA IN FRACTURE HEALING.

by

Mark Browman, M.D.

Research Assistant

National Research Council

Canada

A McGill University thesis submitted to the Faculty of Graduate Studies and Research as partial fulfillment of the requirements for the degree of Master of Science.

August, 1953.

ACKNOWLEDGEMENTS

The past year at the Donner Building has indeed been a very happy and worthwhile experience. It provided the opportunity to become acquainted with the experimental approach to surgical problems, an essential part in the training of the "Complete Surgeon". It also provided pleasant surroundings and the company of a fine group of fellow workers.

To Dr. J.G. Shannon goes a large vote of thanks and appreciation for his helpful suggestions, his keen interest and his invaluable direction throughout the year.

The author is extremely grateful to Dr. D.R. Webster, Director of the Laboratory, who made possible the year at the Donner Building. Dr. Webster in his roll of adviser was superb.

Dr. J.M. McIntyre was especially kind in demonstrating to the author the technique of the operations and in turning over to him data compiled at an earlier date.

Appreciation must be expressed to Dr. J.E. Pritchard and his Laboratory Staff for the preparation of a great number of microscopic sections. Dr. Pritchard was extremely helpful in his interpretation of the many difficult problems which arose in connection with the pathology. Some further sections were prepared by Mr. Hans Torunski.

To the operating room staff goes a vote of praise and thanks. The trio led by Mr. Paul Roustan has been of invaluable service throughout the year. Mr. George Macauley, a good fellow and always willing to lend a helping hand. Mr. Jim Byers capably prepared the animals for operation. In addition to his routine work, Mr. Roustan prepared all the photographs for this manuscript.

The spirit of co-operation which one encountered in the Biochemistry Laboratory was indeed gratifying. A bouquet of roses to Miss Roberta Tyler and her capable assistant Mrs. Virginia Garreau.

Finally a word about the person responsible for a good deal of the success and happiness of the past year - the person who untiringly typed this entire manuscript and who in her typical fashion helped to make the rough going appear smooth. To that person, the author's wife, his sincere thanks.

TABLE OF CONTENT

TABLE OF CHARTS AND ILLUSTRATIONS

A). CHARTS

1). DOG EXPERIMENTS

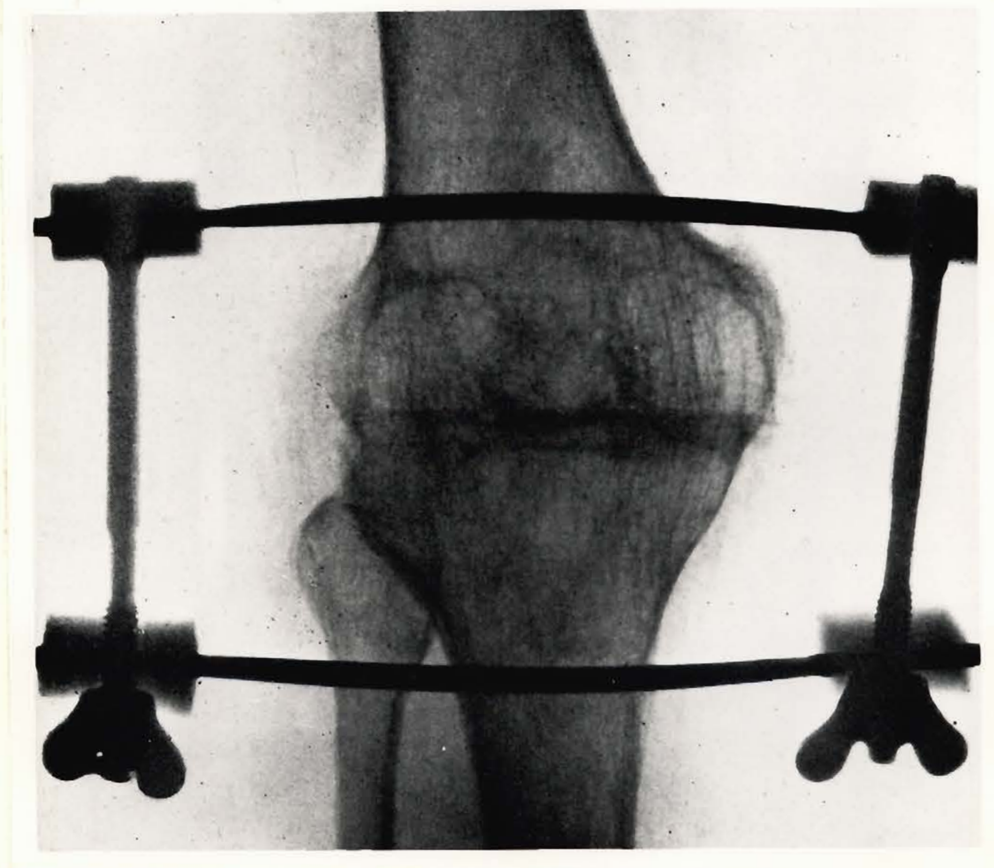
2). RABBIT EXPERIMENTS

B). ILLUSTRATIONS

INTRODUCTION AND REVIEW OF LITERATURE

That distraction of fractured bony surfaces predisposes to delayed union and non union is an accepted principle in Orthopedic Surgery. Every text book on the subject stresses the dangers of distraction and condemns all methods of treatment which may produce separation of the fractured surfaces. Is it not reasonable to assume that the opposite force, namely that of compression of the bony surfaces, would hasten healing? Much work has been done in an effort to prove that a compression force, one that tends to impact the bony surfaces, is valuable in the healing of fractures.

In 1932 Key advocated the use of compression in the arthrodesis of tuberculous knee joints. Using turnbuckles to produce compression he reported union in an unusually short time in 4 of a series of 5 cases. His work went relatively unnoticed for a period of 16 years and then in 1948 Charnley took it up again. Using a similar approach with pressures varying from 60-80 lbs.

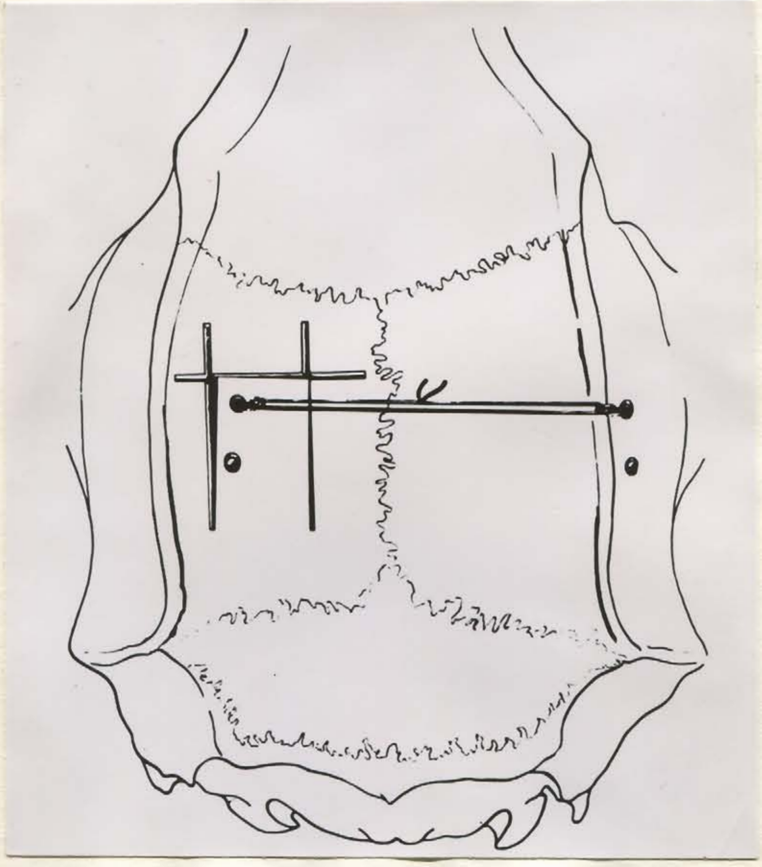


Photograph (#1).- Shows the apparatus used by Key to obtain compression. Charnley, later, used a similar type of apparatus.

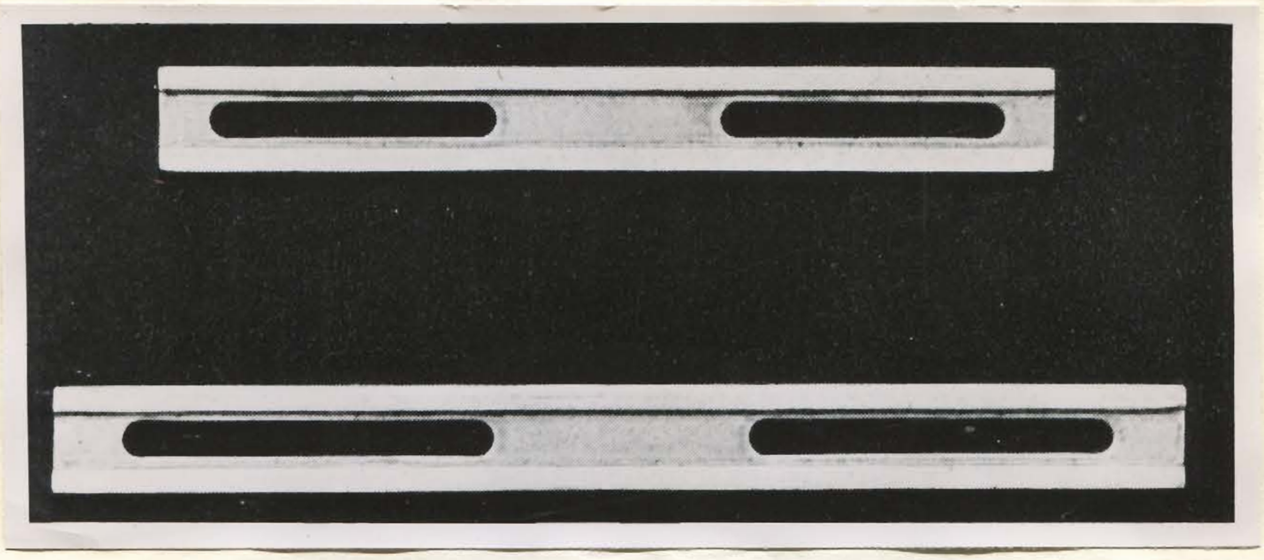
he reported a series of 15 cases in which clinical union was achieved in 4 weeks, bony union in 8 weeks and return to work in 3 months. Thus cutting down the period required for healing by other methods a full 50%.

Eggers fashioned a bone flap in the skull of rats bringing parts of the flap into contact with bone of the skull under compression. He noted that contact compression favourably influenced union. The actual compression force was not measured but he assumed that the most useful force would be very close to the physiolgical force exerted by muscle pull in the area involved. He subsequently devised a slotted bone plate for use in open reduction of fractures to take advantage of the compression exerted by muscle contraction and to insure accurate apposition of the fragments during healing. With this slotted plate the deficiency occuring at the bony ends due to absorption is automatically obliterated.

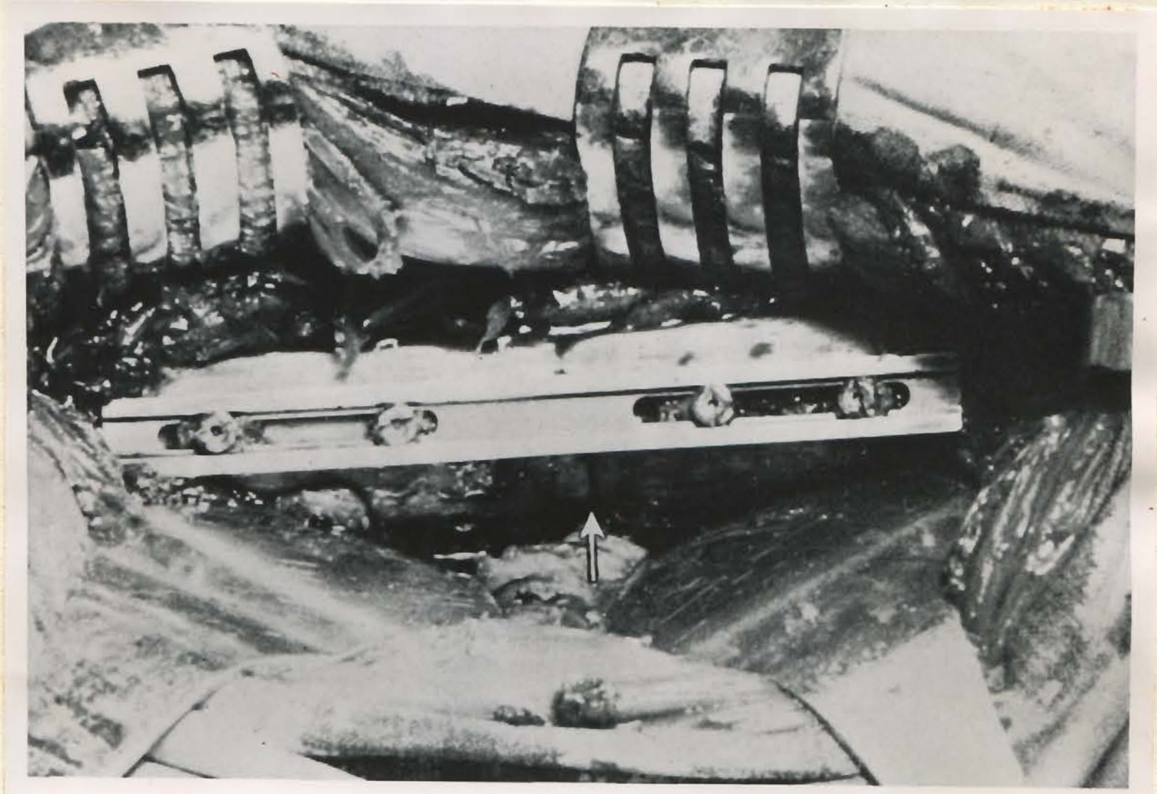
Freidenberg and his associates, assuming that compression plays a beneficial role in fracture healing, set



Photograph (#2).- Shows the bone flap in the skull of rats devised by Eggers.



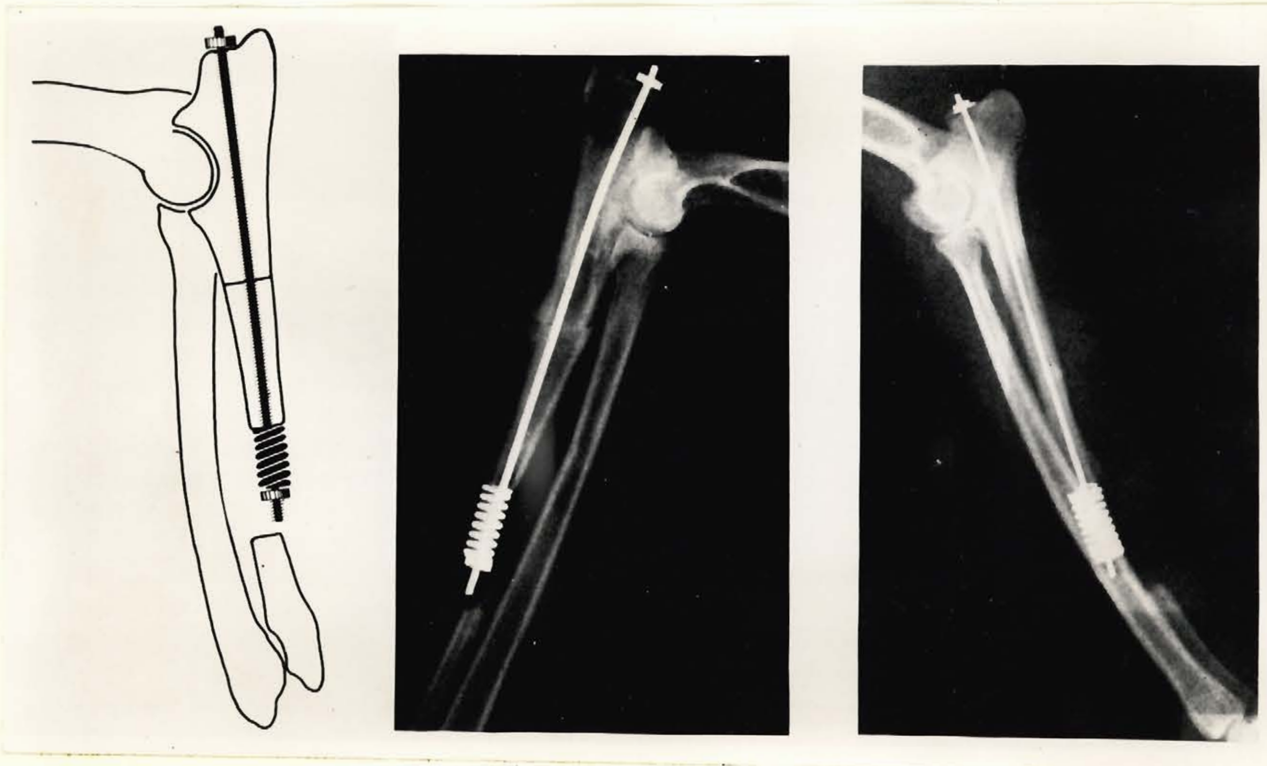
Photograph (#3).- Shows the slotted plate devised by Eggers.



Photograph (#4).- Shows the slotted plate applied across a fracture line.

out to ascertain what pressures would yield the maximum benefit. From their work on the ulnae of dogs it appears that a force of from 12-18 lbs. applied across the fracture line results in more rapid union than in those cases with greater or lesser pressures. Since compression normally exerted by muscle pull is about 6 lbs. Freidenberg concluded that the optimum compressing force is above that which normally results from muscle pull.

It is hard to understand how muscle pull could produce a continous compression force across a fracture line, for once the muscle spasm in response to the fracturing force is spent, compression from this source would automatically cease. At best, such a compression force would be fleeting and short lived. It is also a fact that muscles respond by atrophy to the immobilization which is necessary for fracture healing to occur. Surely such an atrophic group of muscles would produce little in the way of a sustained compression force. Furthermore when a fracture occurs in an atrophic



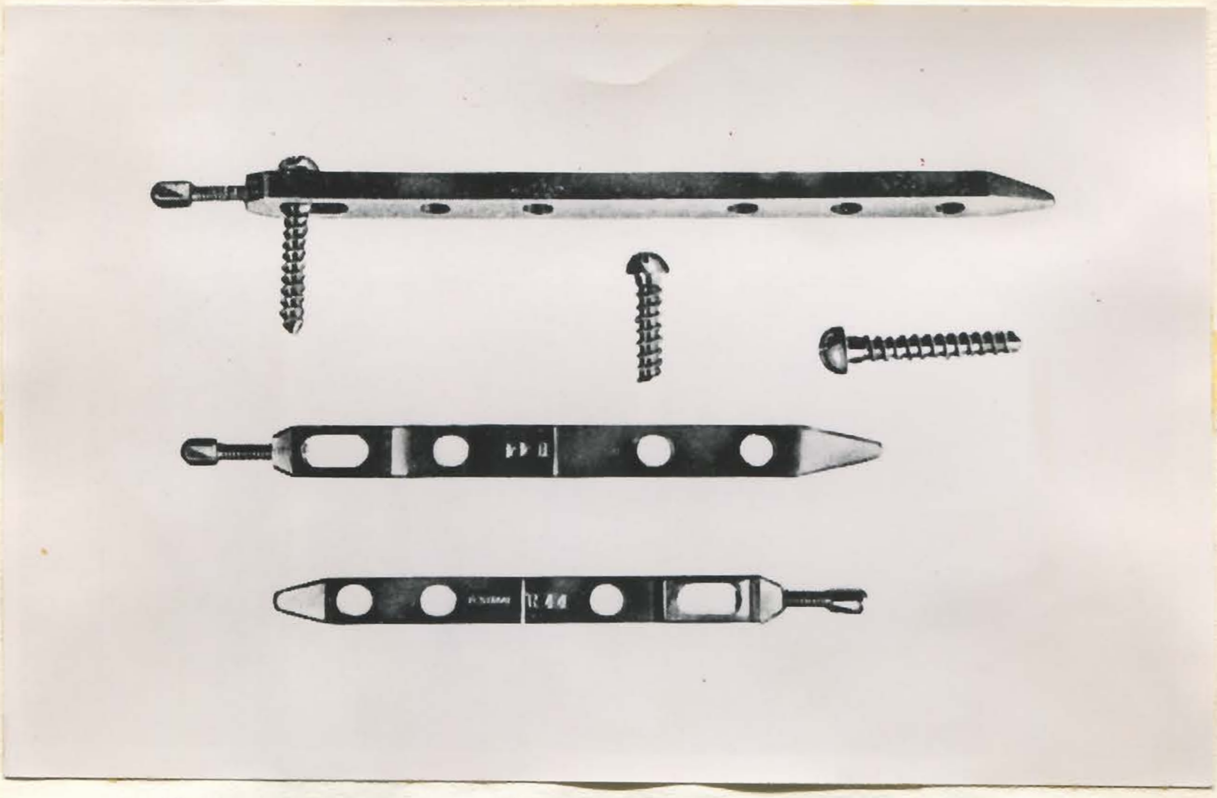
Photograph (#5).- Shows the apparatus and the type of operation employed in the work by Freidenberg.

and wasted limb of a child with poliomyelitis it unites normally in spite of a muscle pull which of necessity would be almost nil.

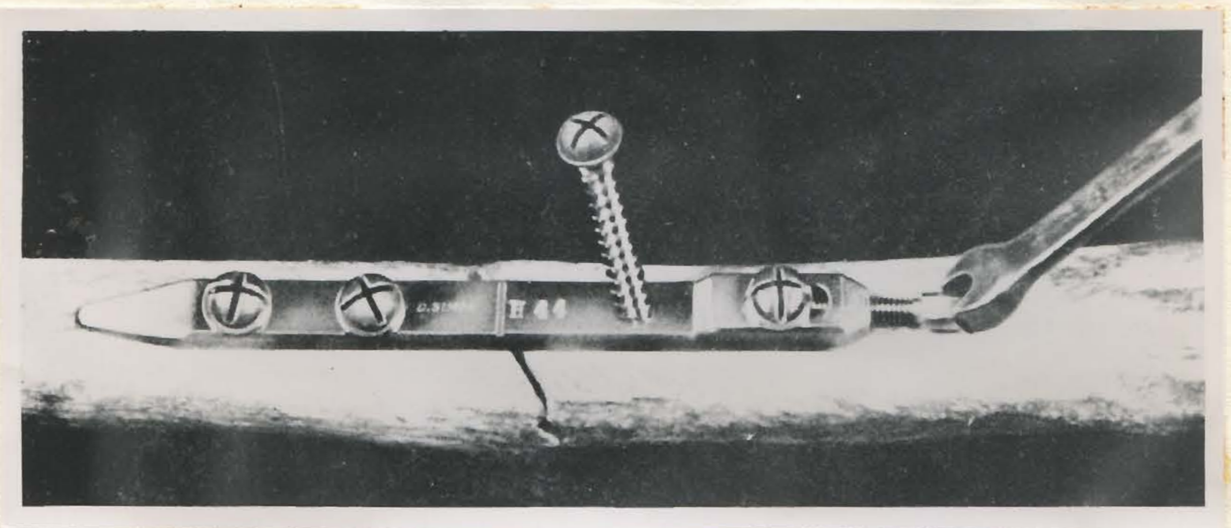
Impressed with the value of compression in fracture healing Henderson and Putti each devised lag screws for the treatment of fractures of the femoral neck. With these screws the head and neck of the femur are firmly impacted. They report encouraging results by this method.

Brittain in his book, "Architectural Principles in Arthrodesis", stresses the value of bone grafts in joint fusion. In describing the method of placing the graft across the joint he states in bold print - "certain elementary principles must be observed, the first is that the graft should be placed with its long axis in compression rather than in tension".

Danis, working in Brussels, designed a plate capable of impacting the fractured surfaces under what he calls axial pressure. He states, "we have taken up



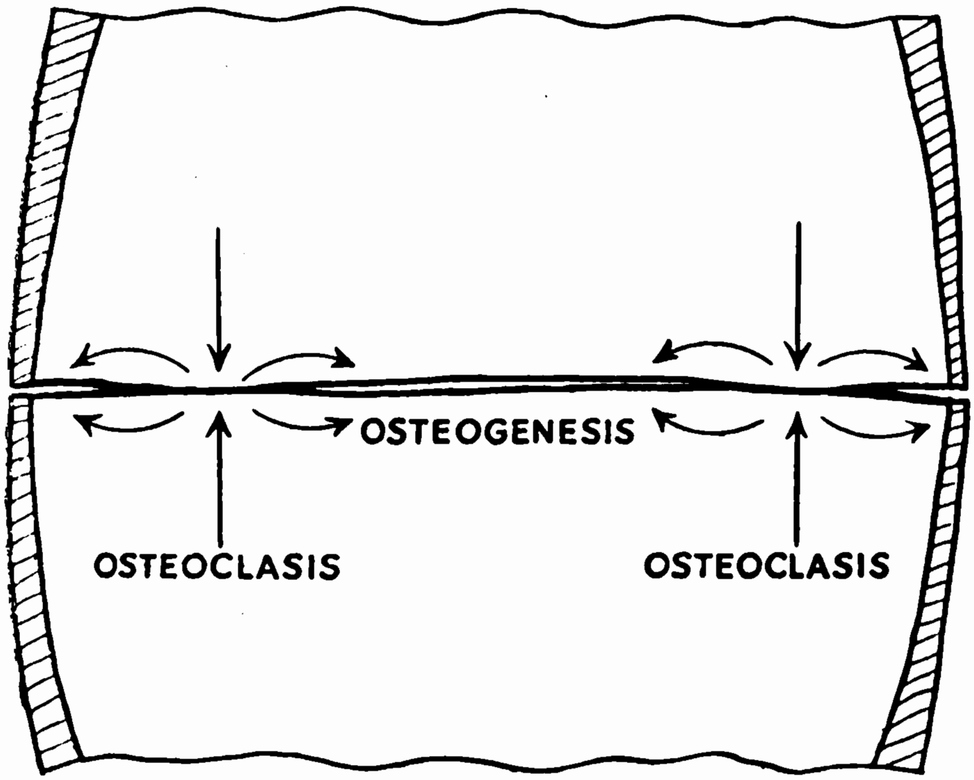
Photograph (#6).- Shows the coaptation splint of Danis.



Photograph (#7).- Shows the coaptation splint being applied across a fracture line.

with a system of plates which after being affixed to the bones can be shortened at will so as to press the fragments firmly together. We call them Coaptation Splints. They made a great change in our results. The healing quality has been greatly improved by the rapid and discrete callus building - quite different from the bulky one you get from the medullar nailing process. I am convinced that the axial pressure is worth much further sound study".

From observations and results such as mentioned above, the thesis that compression is valuable in hastening the healing of fractures gained support. To the compression force was attributed the ability to produce a greater immobility of the fragments and to maintain rigid, prolonged and continued impaction. The more enthusiastic even suggested the possibility that pressure may in itself stimulate osteogenesis at the fracture site. Charnley has put forth an hypothesis in an attempt to explain how pressure may stimulate the healing process. He points out that high compression forces act only on the high



Photograph (#8).- Is a diagramatic representation of the theory presented by Charnley to explain how a compression force may act to stimulate the healing of fractures.

spots between apposed bony surfaces. At these points osteoclasis may be stimulated with consequent absorption of these high spots thus allowing the rest of the bony surface to come into intimate contact. Simultaneously with the removal of bone by osteoclasis the bone substance thus made available may be redeposited by osteoblasts a few millimeters away at points where there is no pressure.

Although the data accumulated seems to indicate that bone formation is encouraged by compression it is by no means accepted by all surgeons engaged in treating fractures.

In 1890, Hugh Owen Thomas wrote, "the practice of jamming the fragments will ere long be found not to be any advantage upon the methods of the past".

Leadbetter feels that positive pressure has very little to do with osteogenesis and points out that impacted fractures of various bones often show a definite absorption line through the area of impaction rather than any increased osteogenesis.

Strickler states that pressure causes bone absorption, interferes with blood supply and with bony union.

Ford and his group, investigating the role of pressure in healing of fractures, used autogenous rib grafts and compared the progress of union of the grafts with and without pressure. The rib grafts were implanted into both iliac bones on one side (the pressure side) the graft was bent and its ends sprung into drill holes. On the control side the graft was placed in drill holes without pressure. These authors concluded that pressure made no difference in the manner and the rate of healing.

Watson-Jones takes definite exception to the idea of compression and in his recent book the discussion of the problem is entitled, "The fallacy of the compression factor in accelerating the union of fractures." He points out that bone resorption occurs in response to pressure, as when a pulsating aneurysm beats against the bones of the spine, or when a meniscus expands and presses firmly on the tibial tuberosity and as when a simple ganglion weighs heavily on the underlying bone.

Phemister advocates simple onlay bone grafts without screw or tie fixation in the treatment of un-united fractures and his work has been quoted time and time again in an effort to belittle the value of a compression force. For here apparently union of bony surfaces is achieved with no more than contact apposition.

To accurately evaluate the data accumulated by the antagonists of the pressure theory, certain points must be considered.

In the case of the experiments performed by Ford and his group, where autogenous rib grafts were used, it should be noted that this work deals with compression of a graft against viable iliac bone whereas in a fracture compression is applied against two viable surfaces. Abbott, in discussing this work, points out that the amount of pressure on the pressure side was inconstant because, as the Authors state, "the ribs lose their elasticity with time".

Watson-Jones stresses the fact that compression of bony surfaces produces bone resorption. It must be remembered that those who advocate compression do not dispute the fact that some resorption does occur at the fracture line and the devices suggested to achieve compression make allowance for the occurance of a degree of resorption. There is, for example, the slotted plate of Eggers which is designed to allow for the occurance of resorption. The fact that pressure produces some resorption of bone does not automatically prove that it also interferes with bony union.

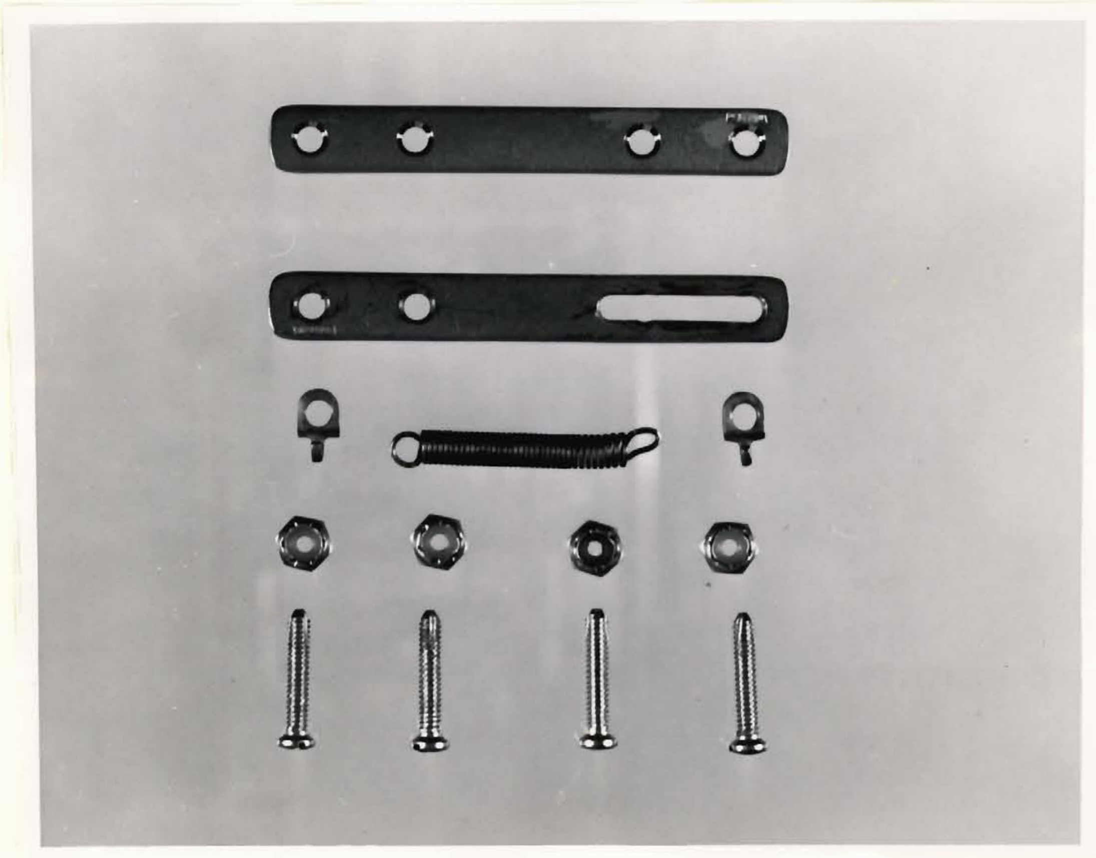
In connection with Phemister's work, if one reads the original article carefully it is noted that he pleads for accurate apposition of the host and graft surfaces and insists that the graft be held firmly against the host bone. It is quite true that here there is no fixation of the graft to the host surface by foreign material, yet it is easy to visualize such a graft firmly compressed against the host bone by a force obtained from the overlying soft tissues and by the application of a snug fitting plaster cast. It would appear that the success of this method is at least in part due to the fact that it allows for firm compression of the apposed bony surfaces.

The delay in union that occurs in lower limb fractures when early weight bearing is allowed has also been used as an argument against compression. It seems unjustified to blame compression for the delayed union that occurs when lower limb fractures are treated by early weight bearing. For in these cases delayed union is more likely due to inadequate immobilization allowing for movement of the fragments and thus damaging the young callus by the intermittent jarring of the fragments against one another during the act of walking. Surely such a compression force, intermittent, uncontrolled and without the certainty of complete immobilization, is by no means the type of compression advocated by those who feel that pressure at the fracture site is useful.

THE EXPERIMENTAL APPLICATION OF A COMPRESSION FORCE ACROSS A FRACTURE

In an effort to study the effects of a compression force across a fracture line an internal splint was designed. This apparatus is able to compress the fractured surfaces together under pressure. It consists of a 3 inch bone plate with a ¾ inch slot at one end and 2 screw holes at the other, a spring made of coiled stainless steel wire .003 inches thick and 1 inch long with a compression force of 15 lbs. when stretched to 1½ inches and special hooked lugs, which fit between the heads of 2 screws and the plate, designed to hold the spring under tension along the long axis of the bone.

The experimental animals were large sized adult dogs. Under intravenous nembutal anaesthesia the femurs of these dogs were exposed and fractured transversely at the mid shaft. The fracture was achieved by placing consecutive drill holes across the mid zone of the bone and then joining these with the use of the osteotome and mallet. The compression apparatus was then applied



Photograph (#9).- Showing the component parts of the compression apparatus employed in our work on dogs.

to the bone. The plate was fixed firmly to the proximal fragment with 2 screws, the screw farthest from the fracture line holding one of the hooked lugs for reception of the spring. Two screws were then inserted into the distal fragment through the slot in the bone plate, care being taken to avoid contact of the screw heads with the plate and to leave about $\frac{1}{4}$ of an inch of space between the proximal screw and the proximal end of the slot. A hooked lug for reception of the other end of the spring was applied to the distal screw in the slot.

Post operatively no external fixation in the form of a cast, splint or caliper was used. The animals reacted rather badly to the restraint offered by such an apparatus and furthermore they generally did not bear any weight on the affected limbs in the early stages, after the operation, because of pain.

Recovery of function was recorded as follows:

No weight bearing......................................0

Slight weight bearing with marked limp.................+

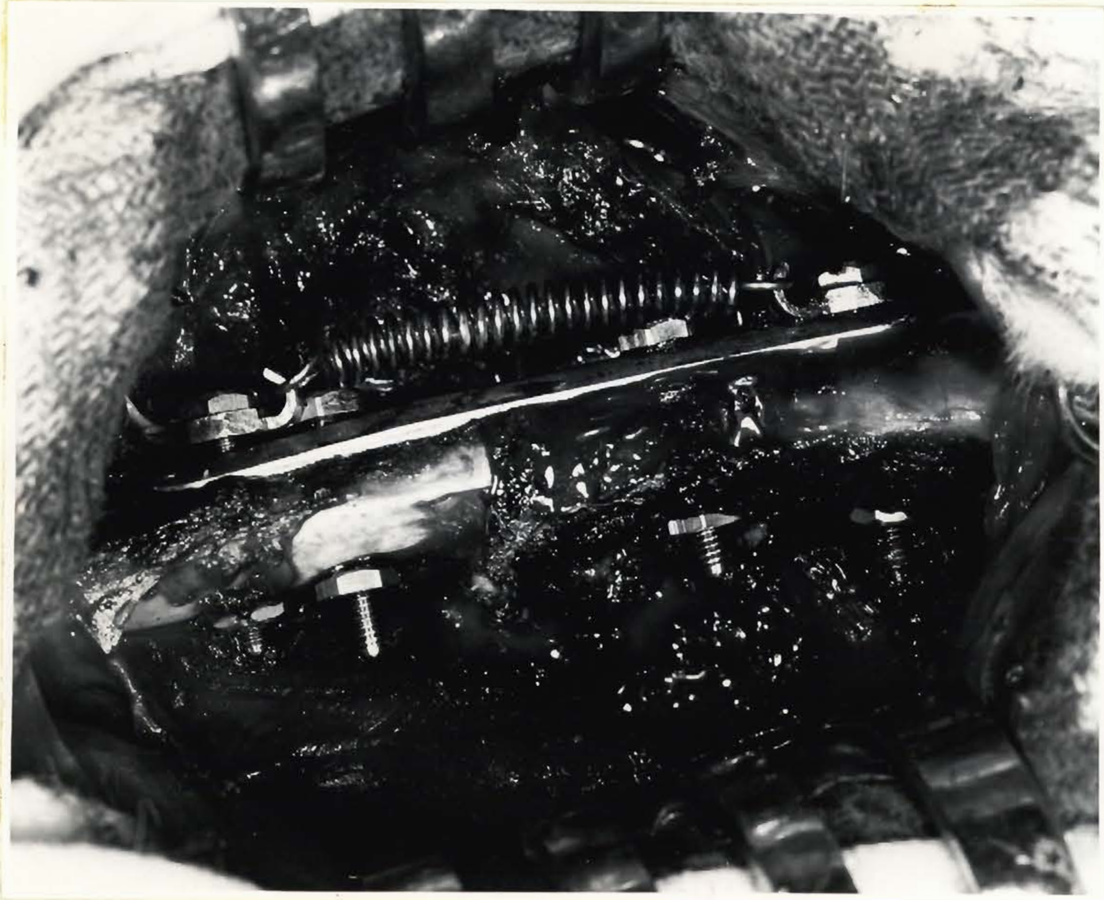
Moderate weight bearing with moderate limp...........+ +

Full weight bearing with slight limp...............+ + +

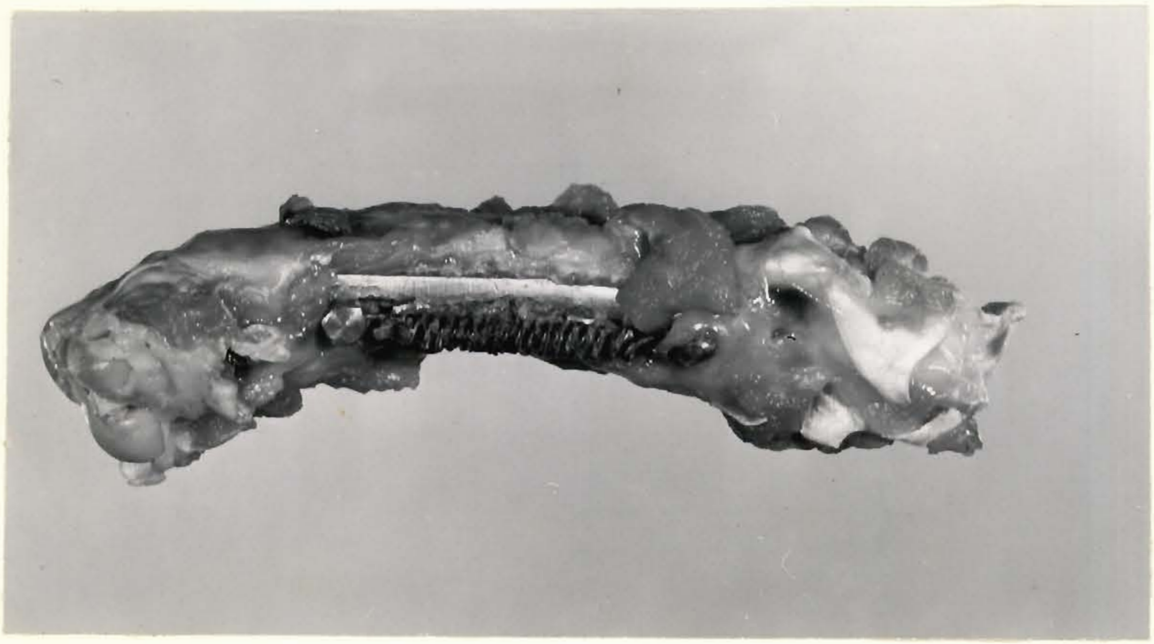
Full weight bearing with no limp.................+ + + +

The progress of union was followed at intervals by x-ray. Animals were sacrificed at varying periods for removal and inspection of the bones in question. Certain of these were then sent to the laboratory to be decalcified, sectioned and stained for microscopic examination.

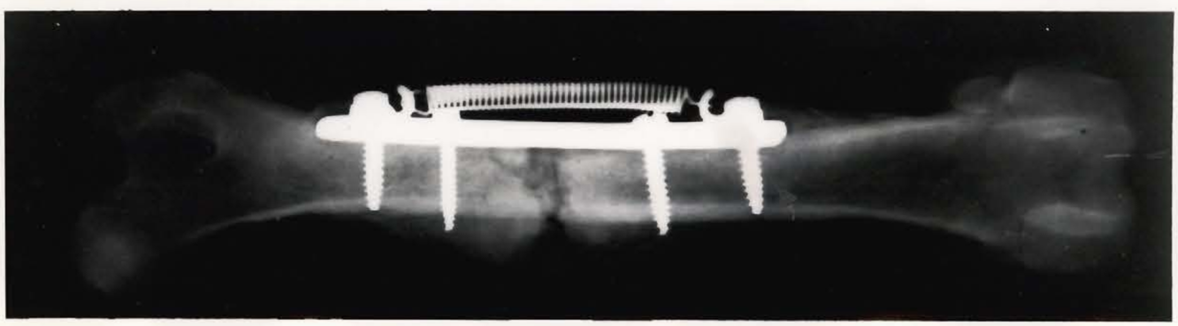
Sixteen fractured femurs in dogs were treated with the compression apparatus. Ten of these fractures went on to complete experiments. From these were obtained fractures of 1, 3, 5, 6, 8, 10, 11 and 20 weeks. The results are charted in table #1. It can be seen from the charts that the recovery of weight bearing function in this group (Group A) is far in advance of the control groups B and C to be described below. Many of these animals were walking with but a slight limp as early as the third post operative



Photograph (#10).- Showing the compression apparatus in place. Note the space between the 2 screws on the left and the plate.



Photograph (#11).- Shows the appearance of the compression apparatus on removal of the bone from the experimental animal.



Photograph (#12).- Is the x-ray of a bone with the compression apparatus applied across the fracture line.

TABLE #1.

GROUP A - FRACTURES OF DOG FEMURS TREATED WITH THE COMPRESSION APPARATUS

DOG#	LEG	DATE OPER'N	# WKS TILL DEATH	TYPE OF OPERATION	CLINICAL OBSER'N / RE WT. BEARING WK1	WK2	WK3	WK4	WK5	WK6	WK7	WK8	X-RAY APPEARANCE	AUTOPSY FINDINGS	PATHOLOGY FINDINGS
2	Left	March 1/51.	10	Single Spring	0	+	++	+++	++++				United.	United.	——
2	Right	May 2/51.	1	Double Spring	0	-	-	-					——	——	——
4	Left	March 15/51.	11	Single Spring	+	++	++	+++	++++				United	United	United.
4	Right	May 16/51.	3	Double Spring	0	+	++						Un-united	Beginning Union	Beginning Union
5	Left	March 28/51.	11	Single Spring	0	+	+	+ +	++	++	++	++	United	United	United
6	Left	March 29/51.	10	Single Spring	0	+	++	+++	++++				"	"	——
7	Left	April 6/51.	20	Single Spring	+	++	+++	+++	++++				"	"	United
7	Right	July 11/51.	8	Single Spring	0	+	++	+++	++++				"	"	United
26	Right	Sept. 24/52.	6	Single Spring	0	++	+++	+++	++++				Advancing Union	"	Advanced Union
28	Left	Oct. 23/52.	5	Single Spring	+	++	+++	++++					"	"	Advancing Union.

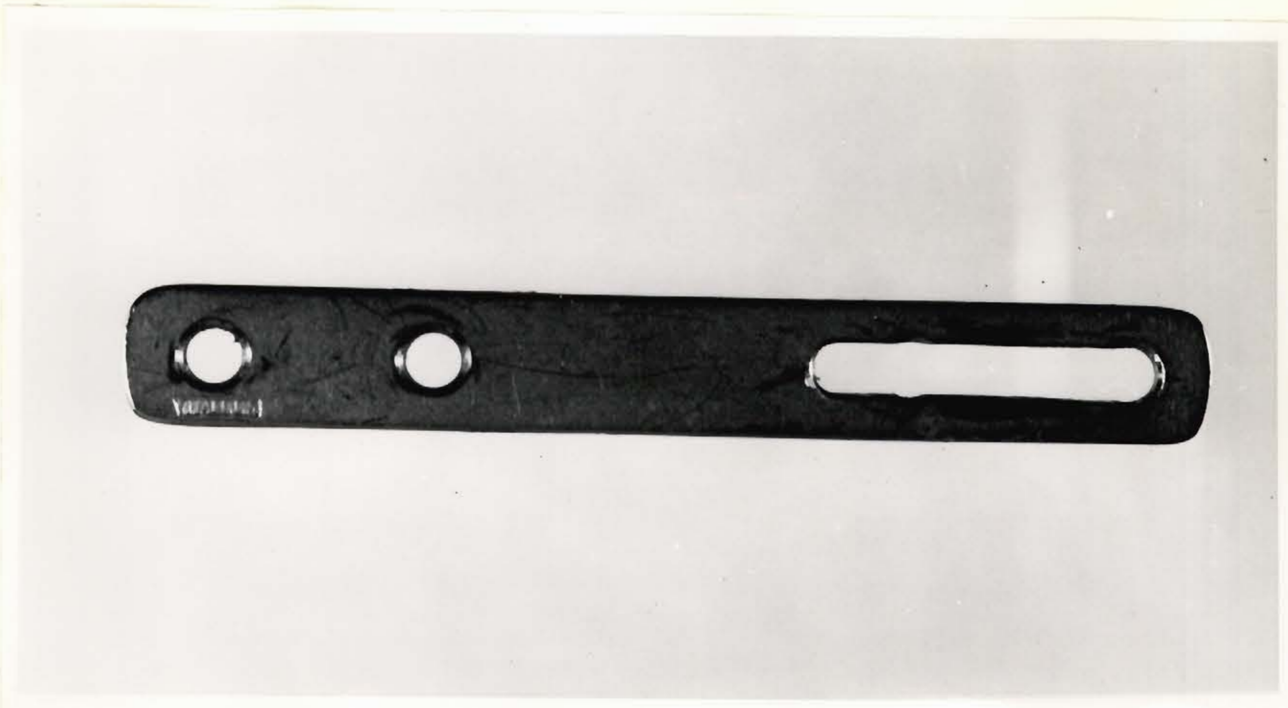
week. By x-ray there is evidence of advanced union after 5 weeks. Photograph (#13) shows the x-ray of femur of dog #28 treated for 5 weeks with the compression apparatus. The fracture site has been reduced to a hair line and bony trabeculae can be seen bridging the gap between the fragments. Union is progressing satisfactorily in this case. Photograph (#14) shows the x-ray of right femur of dog #7 after 8 weeks of treatment with compression. The fracture line has been completely obliterated and there is solid bony union. Photograph (#15) shows the x-ray of the left femur of dog #4 after an 11 week period of compression. Complete bony union has obviously been achieved in this case. Furthermore the restoration of the normal architecture by the process of remodeling, in this group, is occuring more rapidly than in similar fractures of the control groups.

Control experiments were also done. In these cases the bones were fractured, as described above and treated by internal fixation using either a slotted plate without a spring or an intramedullary nail. A total of

Photograph (#13).- Shows x-rays of the femur of dog #28 treated for 5 weeks with the compression apparatus. Union is progressing satisfactorily.

Photograph (#14).- Shows x-ray of the femur of dog #7 treated for 8 weeks with the compression apparatus. Union has occured.

Photograph (#15).- Shows x-ray of the femur of dog #4 treated for 11 weeks with the compression apparatus. Union has occured.



Photograph (#16).- Shows the slotted plate used to immobilize the fractures in one of the control groups. (Group B)

11 control experiments were carried out. Of these, 6 fractures were immobilized using plates without spring compression. From these were obtained fractures of 1, 4, 6, 8 and 9 weeks. The results are charted in table #2 All dogs showed poor recovery of weight bearing function and every dog, regardless of the time he was sacrificed, walked with a marked limp and was able to bear but limited weight on that limb.

Photograph (#17) shows the x-ray of the femur of dog #5. This fracture was treated with a slotted plate for a 4 week period. There is no x-ray evidence of bony union in this case. Photograph (#18) shows the x-ray of the femur of dog #3. This fracture was also treated with a slotted plate for a 9 week period. There is here no x-ray evidence of bony union. At autopsy not one of the bones in this control group (group B) were united.

The remaining 5 fractures were immobilized by the use of intramedullary nails. From these were obtained fractures of 3, 5, 7, 8 and 9 weeks. The results are

TABLE #2.

CONTROL GROUP B - FRACTURES OF DOG FEMURS TREATED WITH BONE PLATES - NO COMPRESSION

DOG#	LEG	DATE OF OPER'N	# WKS. TILL DEATH	TYPE OF PLATE	CLINICAL OBSERVATIONS-RE WT BEARING									X-RAY APPEARANCE	AUTOPSY FINDINGS	PATHOLOGY FINDINGS
					wk1	wk2	wk3	wk4	wk5	wk6	wk7	wk8	wk9			
3	Left	March 8/51.	9	Slotted	0	+	+	+	+	+	+	+	+	Un-United	Un-United	———
3	Right	May 9/51.	1	Non Slotted	0									"	"	———
5	Right	June 6/51.	4	Slotted	0	0	0	+						"	"	Un-United
8	Right	July 20/51.	8	Slotted	0	0	0	0	+	+	+	+		Advancing Union	"	"
8	Left	Aug. 3/51.	6	Slotted	0	0	0	0	0	0				Un-United	"	———
12	Right	Sept. 6/51.	6	Non Slotted	0	0	0	0	0	0				"	"	———

Photograph (#17).- Shows x-ray of the femur of dog #5 treated for 4 weeks with a slotted plate. There is no evidence of bony union.

Photograph (#18).- Shows x-ray of the femur of dog #3 treated for 9 weeks with a slotted plate. There is no evidence of bony union.

charted in table #3. In the post operative period these dogs also showed only limited weight bearing with marked limp (a one plus grading) except for dog #16, this animal took on a two plus grading after 7 weeks. Photograph (#20) shows the femur of dog #24 after treatment with an intramedullary nail for 8 weeks. Photograph (#21) shows the x-ray of the above femur. The fracture line is here still quite evident. At autopsy none of the bones in this group (group C) were united.

In comparing the above results, it is to be noted that the fractures of group A treated with the compression apparatus show, by x-ray, advanced union in 5 weeks and complete bony union by the eight week. The fractures in the control groups B and C treated without compression begin to show evidence of union only after an 8 week period.

Comparision of histological sections of femurs treated with and without compression show certain variations in the healing process of the two groups. These differences

TABLE #3.

CONTROL GROUP C - FRACTURES OF DOG FEMURS TREATED WITH INTRA MEDULLARY NAIL.

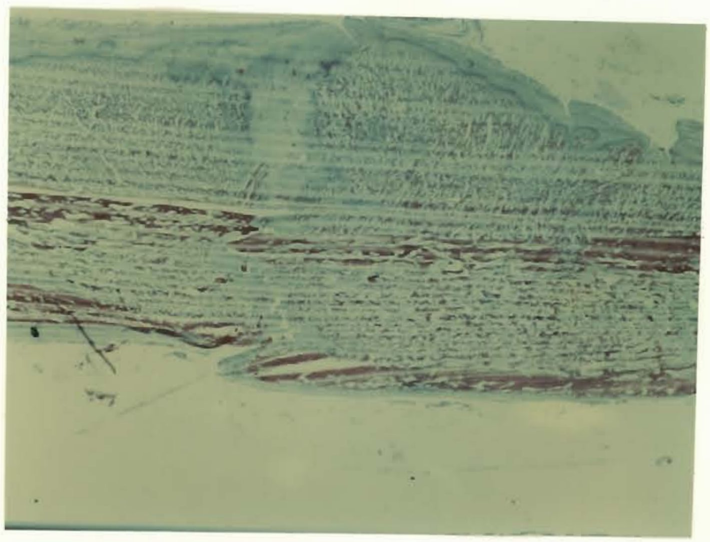
DOG#	LEG	DATE OF OPER'N	# WKS. TILL DEATH	TYPE OF OPER'N	CLINICAL OBSERVATIONS-RE WT BEARING									X-RAY APPEARANCE	AUTOPSY FINDINGS	PATHOLOGY FINDINGS
					wk1	wk2	wk3	wk4	wk5	wk6	wk7	wk8	wk9			
16	Left	May 30/52.	8	Intra Med. Nail	0	0	+	+	+	+	++	++		Advancing Union	Un-United	Advancing Union
17	Left	June 6/52.	7	"	0	0	0	0	0	+	+			——	"	"
19	Left	Aug. 5/52.	3	"	0	0	0							——	"	——
22	Left	Sept. 8/52.	5	"	0	0	+	+	+					——	"	Un-United
24	Left	Sept. 12/52.	9	"	0	0	0	+	+	+	+	+	+	Advancing Union	"	Advancing Union

Photograph (#19).- Shows the intra medullary nail used to immobilize the fractures in the other control group. (Group C)

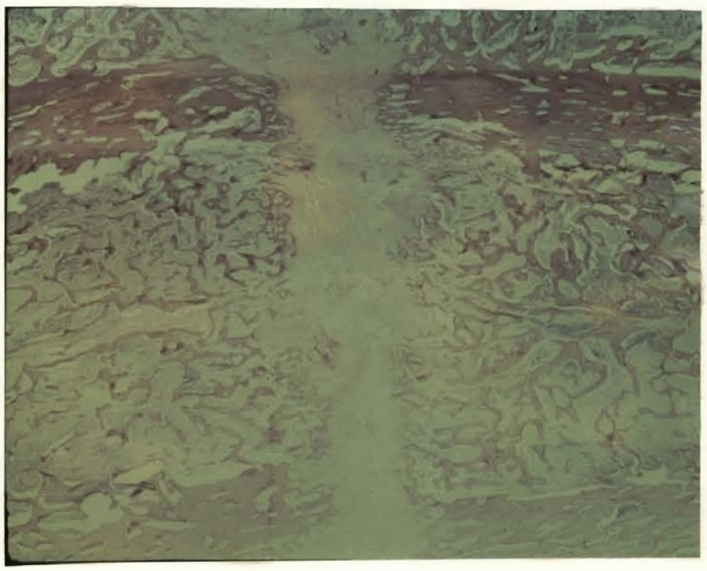
Photograph (#20).- Shows photograph of a fracture treated with intra medullary nail.

Photograph (#21).- Shows x-ray of a femur treated for 7 weeks with an intra medullary nail. Fracture line is still very evident.

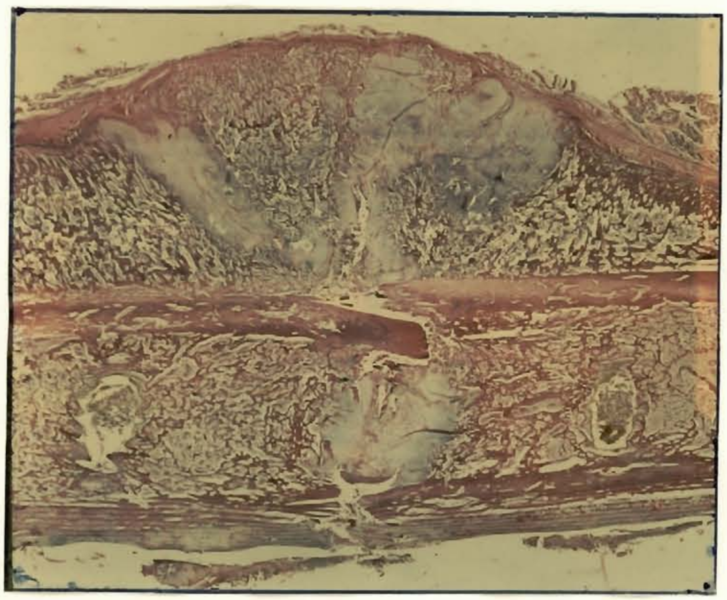
are well demonstrated by sections of the right femur of dog #4 treated by compression for a 3 week period and sections of the right femur of dog #5 treated with a slotted plate without compression for a 4 week period. In both cases there is an obvious paucity of external callus on the side to which the plate was applied. In the former section photograph (#22) the external callus, on the side opposite the plate, is also scanty in amount and dominated by a highly cellular osteoblastic tissue in which bone formation is occuring at a rapid rate. The cartilaginous tissue is here confined to a narrow ribbon in the region of the fracture line. Photograph (#23) shows, with greater magnification, the highly cellular osteoblastic tissue dominating the external callus of a fracture treated with compression. It also shows very well the narrow band of cartilage confined to the region of the fracture line. In the latter section photograph (#24), on the side opposite the plate, there has been formed a massive



Photograph (#22).- Is the histological section of a fracture treated with compression for a 3 week period.



Photograph (#23).- Is a high power study of the previous section.



Photograph (#24).- Is the histological section of a fracture treated with a slotted plate (no compression) for a 4 week period.

amount of external callus which stands out in striking contrast to the previous section. In this section the external and internal calluses are almost entirely composed of hyaline cartilage which extends as a broad sheet for some distance beyond the fracture line.

Bony union is occuring in all these fractures. In the former case the process is dominated by membranous new bone formation with but little cartilage in the region of the fracture line. In the latter case a large amount of cartilage has been formed and the process is predominantly one of endochondral bone formation.

THE ROLE OF THE HEMATOMA IN THE HEALING OF FRACTURES

A fracture is defined as the interruption in the continuity of bone. From the very moment that a fracture is sustained nature sets into motion a series of complex processes aimed at healing the break in bony continuity. The fact that there is no unanimity of opinion as to exactly what happens when a fracture heals is suggestive that there is still much to be learned about this subject.

Let us, for a moment, review a few textbook descriptions of fracture healing as put forth by various authors.

Watson-Jones points out that in the very early stages of fracture healing there is an accumulation of an exudate, which is partly fluid and partly clotted hematoma, between the bone ends and beneath the raised periosteum. Surrounding and invading the hematoma is a rapidly growing loose fibrous tissue of the cellular granulation type. Layers of granulation tissue, formed by each fragment, complete the resorption and organization of the hematoma and if the fragments are immobilized in apposition, both layers meet and unite.

He points out that in the marrow space trabeculae of hypertrophic cartilage cells develop and sometimes almost occlude the medullary cavity. This marks the first stage of callus formation by the endosteum. Similar callus formation takes place beneath the periosteum, especially in young patients where the membrane is easily stripped. The fibroblastic granulation tissue between the fragments, by which continuity has already been established, is then invaded and replaced by the irregular trabeculae of cartilage in which a proportion of bone cells and bone matrix gradually appears.

Hypertrophic cartilage cells form the prominent feature of the histological picture until about the sixth week, but osteoblastic activity and matrix formation continue for a further 3 or 4 weeks. The earlier hyperaemia and vascular engorgement soon disappear. There occurs an increasing calcification and an exuberant growth of a formless, almost tumor-like mass of irregular bone and calcified cartilage which completes this stage of bone union.

Ham's description of the process of fracture healing may be summarized as follows: a few hours after a bone is broken a pool or clot of blood is present in and about the fracture area. It is the result of the blood vessels of the periosteum, bone and adjacent tissue being torn at the time of the injury. The torn vessels bleed from their open ends until they become sealed off by agglutination and coagulation. The swelling of the part caused by the escape of free blood is added to by plasma that leaks through the walls of near-by injured, but not ruptured, capillaries and venules.

Ham emphasizes the fact that bone tissue has cells (osteocytes) scattered throughout its substance in lacunae and that all bone surfaces and the haversian canals are lined with osteogenic cells or osteoblasts.

The cells in bone (osteocytes) and the cells that line the bony surfaces and the haversian canals (osteoblasts) behave very differently after a fracture. The osteocytes near the line of fracture generally die leaving empty lacunae. The lining cells of the periosteum, endosteum and haversian canals undergo a very rapid proliferation. In particular the cells of the inner layer of the periosteum proliferate wildly so that in a short time the osteogenic layer of the periosteum a few millimeters away from the site of the fracture has become many times its original thickness and as a consequence has lifted the fibrous layer of the periosteum away from the bone.

The local thickening of the osteogenic layer of the periosteum of each fragment, a few millimeters away from the line of fracture, is great enough after a few days to have formed an obvious collar around each fragment at this site. Continued proliferation of the osteogenic cells in these two collars causes them to continue to enlarge in size. They expand in two directions: outwardly, and toward the site of the fracture. With time the collars advance toward each other and eventually meet. In this way cellular continuity between the fragments is once more restored.

Proliferation is not the only phenomenon exhibited by the osteogenic cells, for as soon as they begin to proliferate some of them begin to differentiate. Those osteogenic cells, which are closest to the shaft of the fragment where there are capillaries (a vascular area), differentiate into osteoblasts and form trabeculae of bone. As a result of this, the part of each collar that is closest to the shaft of the fragment soon becomes converted into cancellous bone.

The osteogenic cells in the parts of each collar further removed from the shaft, experience a different fate. For differentiating in an area devoid of capillaries (an avascular area) they become chondroblasts and thus form cartilage. This cartilage is later calcified, absorbed and replaced by bone.

Boyd, in his description of fracture healing, points out that when a fracture is sustained there is a tearing of the periosteum and surrounding tissues which results in the pouring out of blood into the area. A certain amount of inflammation also occurs and thus a mixture of blood clot and inflammatory exudate is formed around and between the broken ends of bone.

The exudate is quickly invaded by cells and new capillaries and a kind of granulation tissue is formed. The new cells are osteoblasts derived partly from the deeper layer of the periosteum and partly from the cortical layer of bone. The proliferation of osteoblasts is of an extraordinarily rapid and massive character and there is no other non-malignant process which is quite comparable with it.

In the course of 4-5 days this becomes converted into osteoid tissue, which is tissue resembling bone in its structural arrangement with a homogeneous matrix but not possessing any lime salts. This osteoid tissue or callus becomes increased in amount and acts as an efficient splint. Finally lime salts are laid down and the ends are knit together by fully formed bone.

In the immediate neighbourhood of the fracture the bone cells die. Near the fracture the osteogenic cells proliferate in massive fashion and may form cartilage instead of bone. This cartilage formation is most marked when there is movement or separation of the fragments. The new cartilage is invaded and replaced by bone. This is ossification in cartilage, as compared with the process just described which corresponds to ossification in membrane.

Clay Ray Murray, in discussing the subject of fracture healing, points out that with the fragmentation of bone some local damage to tissue occurs producing tissue death and hemmorage. In response to the autolytic products of tissue death and hemmorage there is the outpouring of an inflammatory exudate into the area. These changes produce stagnation and engorgement of the local minute circulation in the tissue spaces, lymphatics and ultimate capillaries.

The hemmorage and exudate then undergo clotting with the production of a fibrin network. This is followed by the appearance and growth of undifferentiated connective tissue cells along the fibrin network joining the bone ends and the surrounding tissues.

As a result of the accumulation of the products of tissue death and hemmorage the ph of the local tissue fluids is markedly lowered and remains so until such time that the local minute circulation recovers sufficiently to disperse them. The acid ph of the tissue fluids favours the progressive decalcification of bone. The calcium thus made available accumulates in some undetermined form in the connective tissue network.

With restoration of the local minute circulation there is dispersion of the autolysed products of tissue death and hemmorage. Removal of these substances from the fracture area results in a gradual rise in the ph of the local tissue fluids. This is followed by the deposition of the accumulated calcium in the newly formed, undifferentiated, connective tissue network. This is callus formation.

Hence we see that as a fracture heals, there is a procession of less complex tissues leading finally to bone. This procession includes blood clot or hematoma formation, granulation tissue, cartilage (in the region of the fracture line), calcification of the cartilage, absorption of the cartilage, appearance of osteoid and finally calcification of the osteoid.

The role of the hematoma in fracture healing has been stressed by many authors writing on this subject. Watson-Jones, in describing the disadvantages of open reduction of fractures states, "the dissemination and dilution of the fracture hematoma may delay repair".

We wish to put forth a theory of fracture healing which tends to distract from the importance of the hematoma in the healing process.

To serve the functions of weight bearing and support Nature has impregnated a specific soft tissue with calcium salts thereby forming a rigid tissue which is called bone. Bone is, therefore, fundamentally a soft tissue composed of an interlacing network of living cells. These cells line the haversian systems and are condensed on the inner and outer surfaces of the bone into a more regular linear pattern forming the endosteum and periosteum. This soft tissue network is endowed with osteogenic properties. In the following description we shall refer to this tissue, collectively, as the osteogenic tissue.

When a fracture is sustained hemmorage and exudation does occur at the fracture site - but the osteogenic tissue is not raised from the bone, it is stimulated to proliferate. This proliferation takes place very rapidly and as it grows laterally from the shaft it pushes the hematoma before it. The hematoma between the fragments cannot be pushed away and therefore acts as a block to the growth of the osteogenic tissue along the long axis of the bone in the region of the fracture.

It may be that if the hematoma can be evacuated and prevented from reforming without substituting another substance which would block the growth of this osteogenic tissue, then these vital bone forming cells from one fragment would unite directly with similar cells of the other fragment and bone trabeculae would be formed in this tissue directly without the intermediate stages of endochondral bone formation.

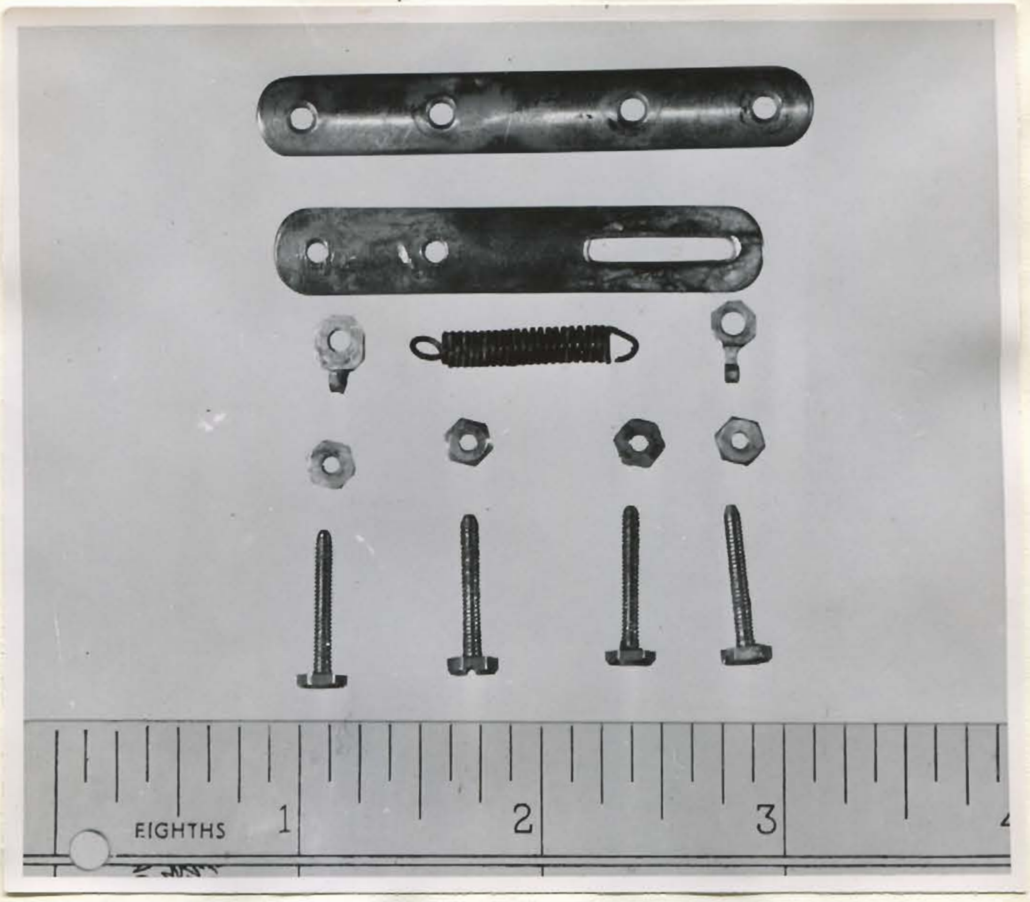
A similar situation exists in the healing of a skin wound. If the edges are well approximated proliferation of fibroblasts and epithelial cells occur from both sides - these soon meet and healing is achieved. If, however, a gap is produced by loss of tissue substance, healing is delayed, the existing defect must first be filled in by granulation tissue which is then converted into fibrous tissue, only then can the epitheluim cover the surface - healing is again achieved. Thus in wound healing the direct approach to repair is impeded by the loss of tissue substance - which substance must be replaced by granulation tissue before the final stages of epithelialization can occur. Similarly in fracture healing the direct approach to repair may be impeded by the presence of the hematoma between the bone ends acting as a barrier to the proliferating osteogenic cells and preventing them from bridging the gap directly. The value of the hematoma is therefore questioned.

In an attempt to test our theory, certain experiments were devised in which the hematoma was evacuated and replaced with a homogeneous structureless mass which would offer no barrier and require no organization or replacement by the rapidly growing osteoblastic cells. Rigid immobilization was then obtained by the use of a compression splint.

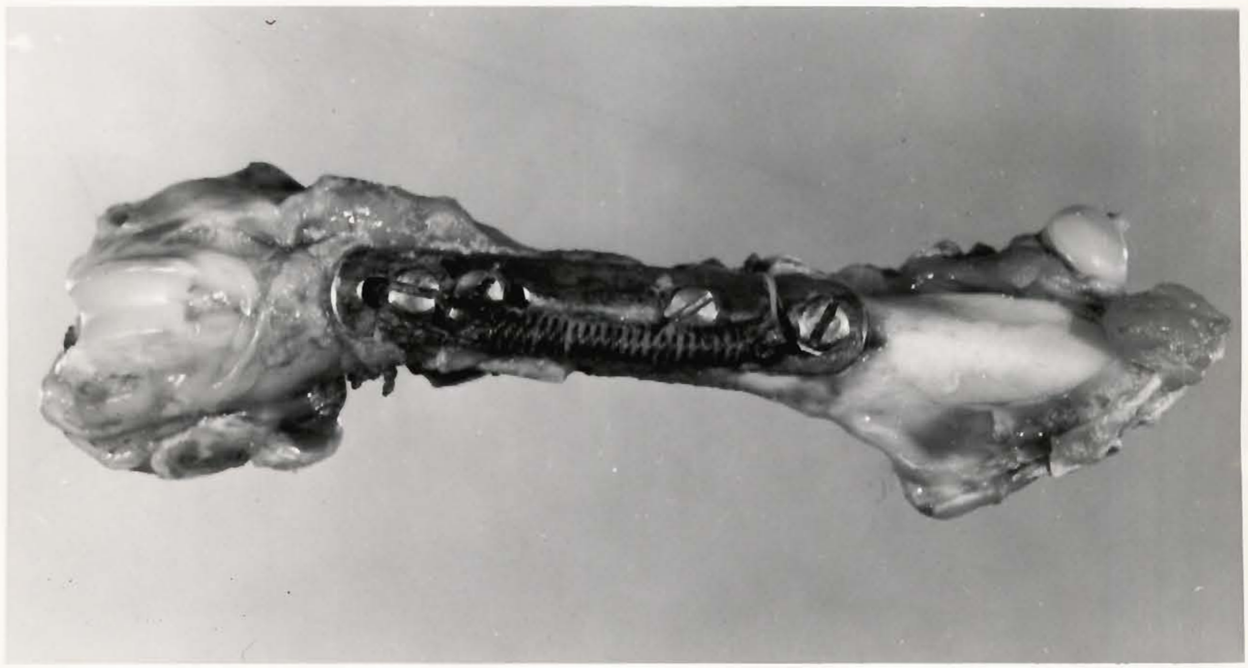
Plasma was the first substance considered to replace the hematoma but it was soon realized that a plasma solution which is able to clot contains fibrin and has a composition closely akin to whole blood. A new substance was sought and gelatine was chosen. Gelatine is a protein and has the property of taking up water to become a homogeneous gel. The gelatine was sterilized in a rubber stoppered flask and introduced, about the fracture site, in its original powdered form.

For this group of experiments large sized adult rabbits were used. The operation on the femur was similar in detail to the one previously described for the dog experiments. The apparatus of stainless steel was also identical but on a smaller scale to conform to the size of the rabbit femur. Bolts were applied to all screws to prevent them from being pulled out by the force of the spring. The coiled spring was calibrated so that when stretched to 1 inch it produced a compression force of 8 lbs. Photograph (#25) shows the apparatus employed in these experiments. Photograph (#26) shows the appearance of the compression apparatus on removal of the bone from the experimental animal. Photograph (#27) shows x-ray of apparatus in place.

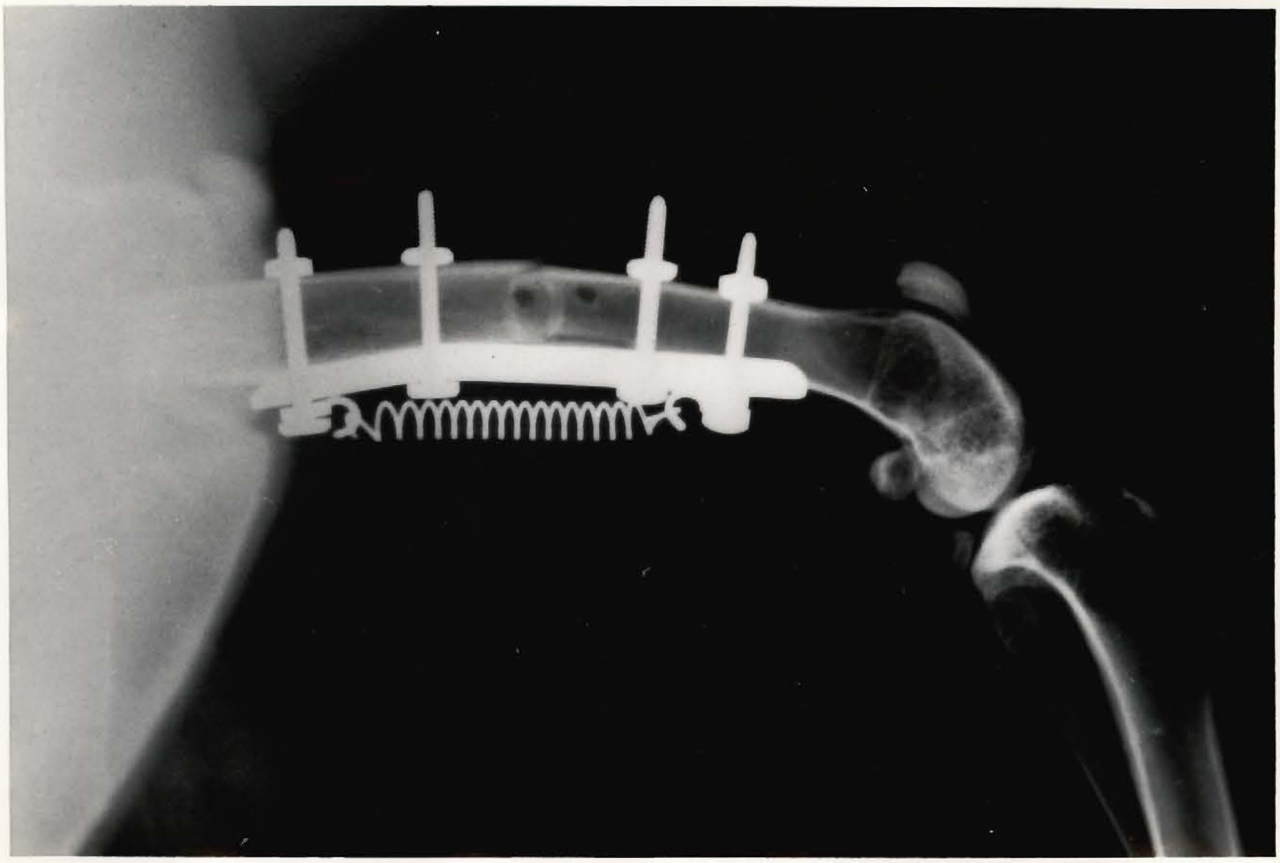
After the fracture was produced in a manner previously described and the compression apparatus applied across the fracture site, the wound was thoroughly irrigated with normal saline. It was then aspirated and dry packs were left in place for from 3-5 minutes to further dampen the small bleeding points. When the area, in the region of the fracture, was completely dry and free from blood, a rubber cuff was applied about the bone and held in place by Allis Forceps at either end. The powdered gelatine was now introduced, in small quantities, into the cylindrical opening in the rubber cuff. Water was



Photograph (#25).- Show the component parts of the compression apparatus employed in our work on rabbits.



Photograph (#26).- Shows the appearance of the compression apparatus on removal of the bone from the experimental animal.

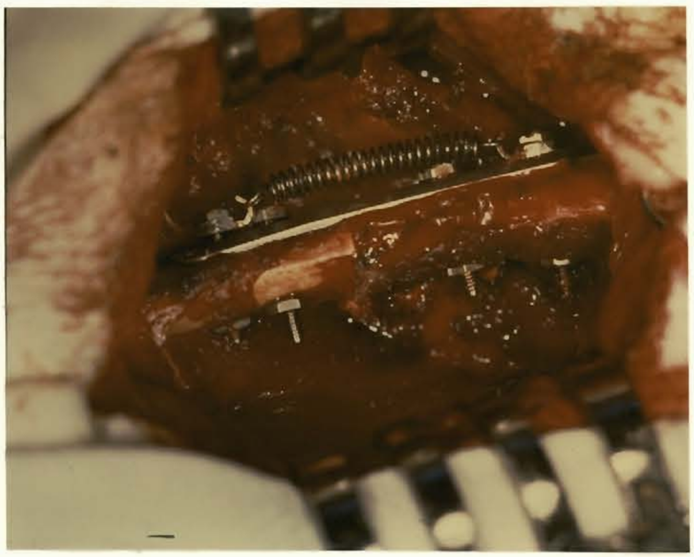


Photograph (#27).- Is the x-ray of a bone with the compression apparatus applied across the fracture line.

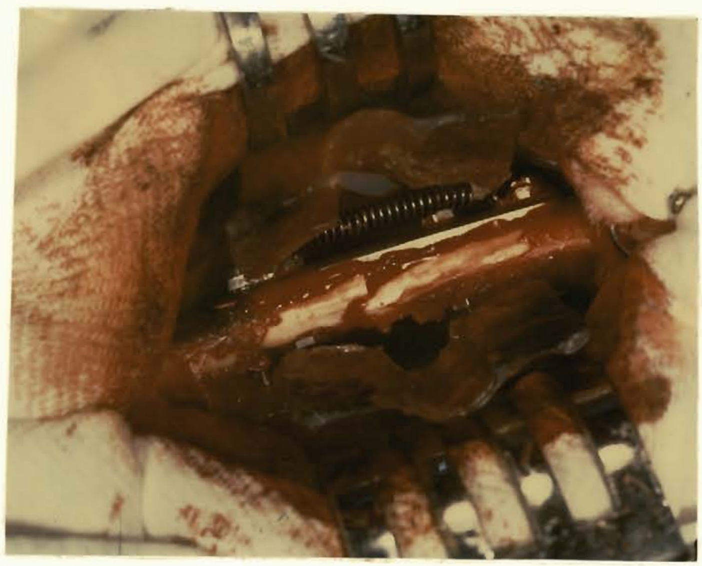
added at intervals and in a few moments a homogeneous gel was obtained. When the entire circumference of the bone, at the fracture site, was enveloped by the gel the rubber cuff was gently removed. The clot was then trimmed so that its extent was limited to about $\frac{1}{8}$ of an inch on either side of the fracture line. The wound was then closed in layers. No external support was applied. Photographs (#28-32) show, in series, the stages of the above proceedure.

As time went on the technique was altered slightly - the rubber cuff about the bone was abandoned for it was found that on removing it, at the conclusion of the experiment, the clot was often disturbed. In later work the gelatine was closely applied to the fracture site with the finger. In this way the powder could be placed more accurately between the fractured surfaces and there was no disturbance of the clot once it was formed. Photograph (#33) demonstrates the above described method of gelatine application.

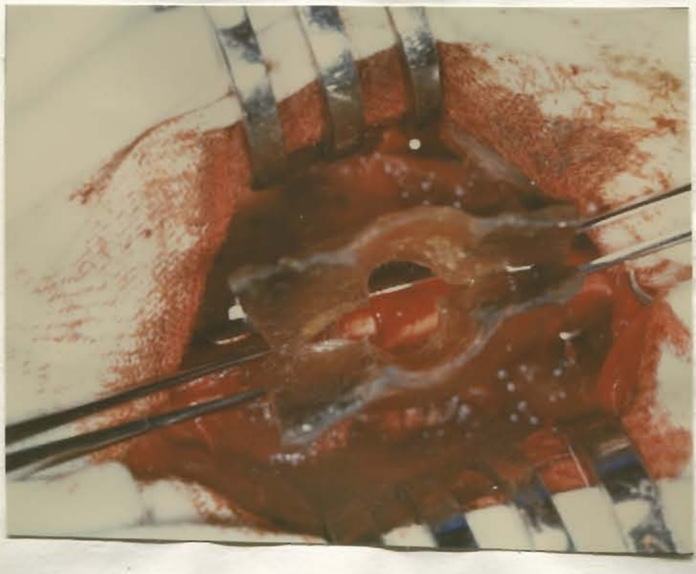
Photographs (#28-31).- Show the stages in the evacuation of the hematoma and replacement of it by gelatine.



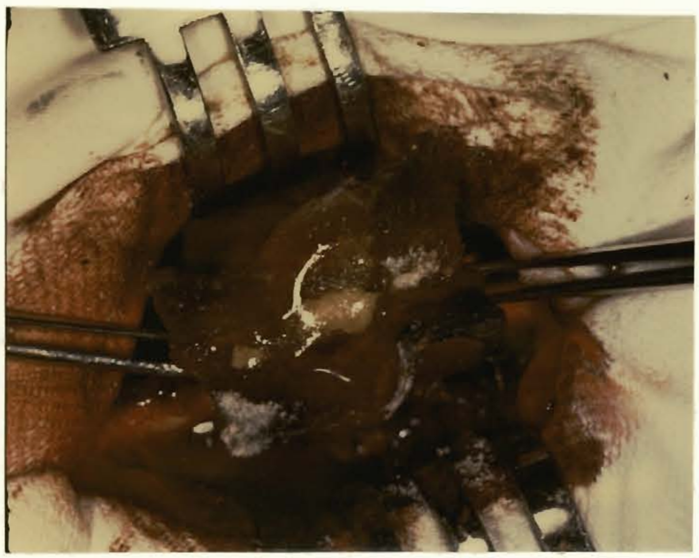
Photograph (#28).- Shows the compression apparatus applied to the fractured bone.



Photograph (#29).- Shows the rubber cuff in place about the bone in the region of the fracture. The hematoma has been evacuated.



Photograph (#30).- Shows the edges of the rubber cuff held tightly approximated with Allis Forceps, thus leaving a cylindrical opening in the cuff in the region of the fracture.



Photograph (#31).- Shows the gel, obtained after adding water to the gelatine which was placed about the fracture line. The gel is contained in the rubber cuff.

Photograph (#32).- Shows the final stage of the process, the rubber cuff has been removed and the gel trimmed so that it extends for about an $\frac{1}{8}$ of an inch on either side of the fracture line.

Photograph (#33).- Shows the method of applying the gelatine to the fracture line with the finger.

The only clinical observations possible on rabbits were those pertaining to the healing of the wound and the testing of the fracture site for union. Progress of bony union was followed at varying intervals by x-ray and femurs were removed, from time to time, for gross examination. In every case it was noticed that the distal screw in the slot had moved proximally, proving that compression had been effective and that some bone resorption had occurred at the fracture line (this was also observed in the dog experiments). Photographs (#34-35) show the above phenomenon. The springs although covered by a layer of thin fibrous tissue contracted to almost their original length on removal.

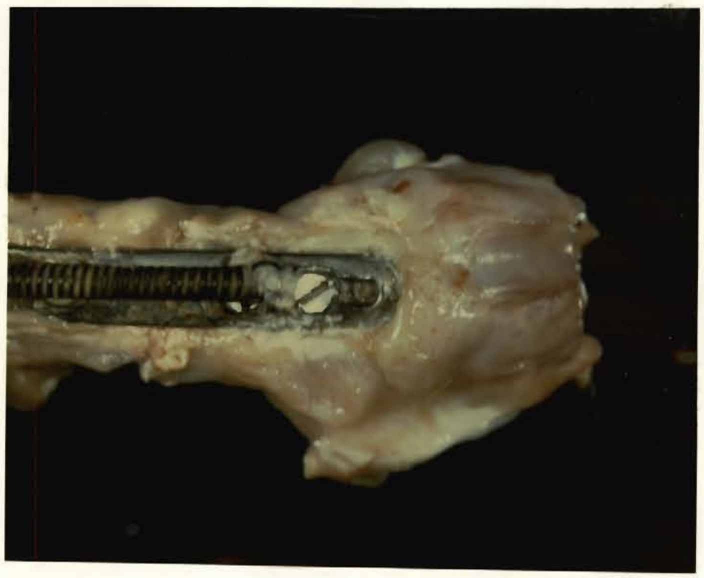
Femurs were then sent to pathology for fixation, staining and sectioning.

Control groups were also kept. These included;

1). Group B - fractures treated with compression, hematoma intact.

2). Group C - fractures treated with bone plate, hematoma intact.

Photograph (#34).- Shows the effect of the compression force by the movement, toward the fracture line, of the distal screw in the slot.



Photograph (#35).- Is an enlargement of the previous picture to show more clearly the movement toward the fracture line, of the distal screw in the slot.

3). Group D - fractures treated with intra medullary nail, hematoma intact.

Photograph #36 shows the type of intra medullary nail used in control Group D.

Table #4, charts the results obtained in experiments using the compression apparatus with evacuation of the hematoma and replacement of it by gelatine. Tables 5, 6 and 7 chart the results obtained in the control groups.

Careful study of the gross specimens, the x-ray films and the histological sections revealed that evacuation of the hematoma and replacement of it by gelatine did not have any effect on hastening the healing of fractures so treated when compared with the control groups. The evacuation of the hematoma and replacement of it by gelatine did not however retard the healing of fractures so treated.

These experiments tend also to confirm the value of compression in the healing of fractures.

TABLE #4.
GROUP A.
FRACTURES OF RABBIT FEMURS TREATED WITH COMPRESSION APPARATUS - HEMATOMA EVACUATED AND REPLACED BY GELATINE.

RABBIT#	LEG	DATE OF OPER'N	# WKS TILL DEATH	X-RAY	AUTOPSY FINDINGS	PATHOLOGY
9	Left	24/52	1	Un-united	Un-united	Un-united
22	"	16/53	2	Un-united	Beginning Union	Un-united
18	"	Jan. 5/53.	3	Un-united	Beginning Union	Un-united
23	"	Jan. 19/53	4	Advancing Union	Advanced Union	1 cortex-union Prog. union along other
33	"	Feb. 6/53.	5	Advancing Union	Union	1 cortex-union Prog. union along other
35	"	Feb. 1 2/53.		Un-united	Union	Fibrous Union

TABLE #5.

CONTROL GROUP B.

FRACTURES OF RABBIT FEMURS TREATED WITH THE COMPRESSION APPARATUS - HEMATOMA INTACT.

RABBIT#	LEG	DATE OF OPER'N	# WKS. TILL DEATH	X-RAY	AUTOPSY FINDINGS	PATHOLOGY
2	Left	Sept. 18/52.	1	Un-united	Un-united	Un-united
29	"	Feb. 3/53.	2	Un-united	Un-united	Un-united
3	"	Oct. 28/52.	3	Early union	Un-united	Un-united
30	"	Feb. 3/53.	4	Advancing Union	Un-united	Advancing Union
31	"	Feb. 9/53.	5	United	United	United
37	"	Feb. 13/53.	6	United	United	United

TABLE #6.
CONTROL GROUP C.
FRACTURES OF RABBIT FEMURS TREATED WITH BONE PLATE - HEMATOMA INTACT.

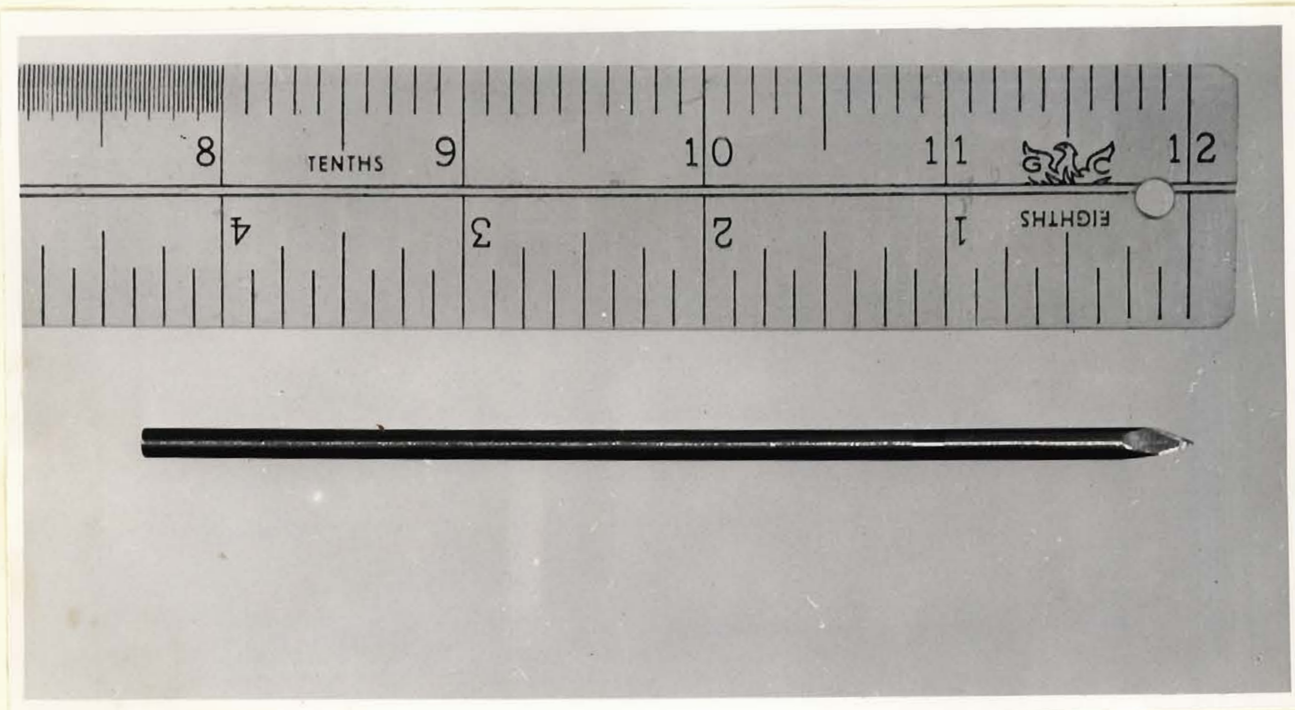
RABBIT#	LEG	DATE OF OPER'N	# WKS. TILL DEATH	X-RAY	AUTOPSY FINDINGS	PATHOLOGY
25	Left	Jan. 22/53.	1	Un-united	Un-united	Un-united
24	"	Jan. 21/53.	2	Un-united	Un-united	Un-united
28	"	Jan. 30/53.	3	Un-united	Early sticking	Un-united
38	"	Feb. 18/53.	4	Un-united	Early sticking	Un-united
43	"	March 5/53.	5	Advancing Union	Beginning Union	Advancing Union
10	"	Nov. 22/52.	6	United	Union	United

TABLE #7.

CONTROL GROUP D.

FRACTURES OF RABBIT FEMURS TREATED WITH INTRA MEDULLARY NAIL - HEMATOMA INTACT.

RABBIT#	LEG	DATE OF OPER'N	# WKS. TILL DEATH	X-RAY	AUTOPSY FINDINGS	PATHOLOGY
20	Left	Jan. 13/53.	1	Un-united	Un-united	Un-united
21	"	Jan. 13/53.	2	Un-united	Un-united	Un-united
16	"	Dec. 15/52.	3	Early union	Beginning union	———
15	"	Dec. 16/52.	4	Advancing Union	Advancing union	Advancing union
26	"	Jan. 29/53.	5	Solid union	Union	Advanced union
32	"	Feb. 6/53.	6	Advanced Union	Union	Advanced union



Photograph (#36).- Shows the type of intra medullary nail employed in our work on rabbits.

SUMMARY AND CONCLUSIONS

To test the hypothesis that compression hastens the healing of fractures, three groups of experimental fractures in dogs have been devised. The first group was treated by plate immobilization and compression. The control groups were immobilized with bone plates or intra medullary nails.

1). It was found that union occured more rapidly in the group treated with compression.

To evaluate the role of the hematoma in the healing of fractures, three groups of experimental fractures in rabbits have been devised. The first group was treated by plate immobilization and compression with evacuation of the hematoma and replacement by gelatine. One control group was treated by plate and spring immobilization with hematoma intact. The other groups were immobilized with bone plates or intra medullary nails without compression and with hematoma intact.

2). Evacuation and replacement of the hematoma with gelatine did not hasten the healing of fractures when compared with the control groups in which the hematoma was left intact.

3). Healing did nevertheless occur in the group treated by evacuation and replacement of the hematoma with gelatine.

4). Fractures treated with compression did show more rapid healing than those treated without compression. It is estimated from this work that compression cuts down the time required for healing by approximately 25%.

BIBLIOGRAPHY

Ainsworth, W.H., and Wright, N.E.
Experimental study of fracture sites.
J. Bone Surg., 30A: 48, 1948.

Bick, E.M.
Structural patterns of callus in fractures of the long bones.
J. Bone Surg., 30A: 141, 1948.

Boyd, W.
Surgical Pathology. 6th ed. W.B. Saunders Co.
Philadelphia, 1947.

Brittain, H.A.
Architectural Principles in Arthrodesis. E & S Livingstone.
Edinburgh, 1942.

Charnley, J.C.
Positive pressure in arthrodesis of the knee joint.
J. Bone Surg., 30B: 478, 1948.

Charnley, J.C., and Baker, S.L.
Compression arthrodesis of the knee.
J. Bone Surg., 34B: 187, 1952.

Danis, R.
Operative treatment of bone fractures.
J. Int. Chir., 7: 311, 1947.

Eggers, G.W.N.
Internal contact splint.
J. Bone Surg., 30A: 40, 1948.

Eggers, G.W.N., Ainsworth, W.H., Schindler, T.O. and Pomerat, C.M.
Clinical significence of the contact-compression factor in bone surgery.
Arch. Surg., 62: 647, 1951.

Eggers, G.W.N., Schindler, T.O. and Pomerat, C.M.
The influence of the contact-compression factor on osteogenesis in surgical fractures.
J. Bone Surg., 31A: 693, 1949.

Ely, L.W.
Experimental study of the healing of fractures.
Arch. Surg., 5: 527, 1922.

Ford, L.T., Lottes, J.O. and Key, J.A.
Experimental study of the effect of pressure on the healing of bone grafts.
Arch. Surg., 62: 475, 1951.

Freidenberg, Z.B. and French, G.
The effects of known compression forces on fracture healing.
Surg. Gyn. Obst., 94: 743, 1952.

Ham, A.W.
A histological study of the early phases of bone repair.
J. Bone Surg., 12: 827, 1930.

Ham, A.W.
Histology. J.B. Lippincott Co.
Montreal, 1950.

Key, J.A.
Positive pressure in arthrodesis for tuberculosis of the knee joint.
South. Med. J., 25: 909, 1932.

Key, J.A. and Reynolds, F.C.
Contact splints (Eggers) vs. standard bone plates in the fixation of experimental fractures.
Ann. Surg., 137: 911, 1953.

Murray, C.R.
Healing of fractures.
Arch. Surg., 29: 446, 1934.

Phemister, D.B.
Treatment of ununited fractures by only bone grafts without screw or tie fixation and without breaking down of the fibrous union.
J. Bone Surg., 29: 946, 1947.

Pritchard, J.J.
Repair of fractures of the parietal bone in rats.
J. Anat., 80: 55, 1946.

Tarlov, I.M.
Plasma clot suture of peripheral nerves and nerve roots. C.C. Thomas.
Illinois, 1950.

Urist, M.R. and Johnston, R.W.
Calcification and ossification - healing of fractures in man.
J. Bone Surg., 25: 375, 1948.

Watson-Jones, R.
Fractures and joint injuries. 4th ed. E. & S Livingstone.
Edinburgh, 1952.

Watson-Jones, R. and Roberts, R.E.
Calcification, decalcification and ossification.
Brit. J. Surg., 21: 461, 1934.